http://www.cas.org/ONLINE/UG/regprops.html

```
=> s inosine/cn
L1            1 INOSINE/CN

=> sel L1
E1 THROUGH E27 ASSIGNED

=> file caplus medline
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         5.53       5.74

FILE 'CAPLUS' ENTERED AT 16:35:10 ON 01 MAY 2006
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FILE 'MEDLINE' ENTERED AT 16:35:10 ON 01 MAY 2006

=> s E1-E27
L2         20622 (ATOREL/BI OR HXR/BI OR "HYPOXANTHINE RIBONUCLEOSIDE"/BI OR
                  "HYPOXANTHINE RIBOSIDE"/BI OR "HYPOXANTHINE 9-B-D-RIBOFURAN
                 OSIDE"/BI OR "HYPOXANTHINE, 9-B-D-RIBOFURANOSYL-"/BI OR
                 HYPOXANTHOSINE/BI OR INO/BI OR INOSIE/BI OR INOSINE/BI OR "NSC
                 20262"/BI OR OXIAMIN/BI OR PANHOLIC-L/BI OR RIBONOSINE/BI OR
                 SELFER/BI OR TROPHICARDYL/BI OR "1,9-DIHYDRO-9-B-D-RIBOFURAN
                 OSYL-6H-PURIN-6-ONE"/BI OR 12712-98-0/BI OR 132953-54-9/BI OR
                 28861-88-3/BI OR 292853-81-7/BI OR 4181-51-5/BI OR 58-63-9/BI OR
                 "6H-PURIN-6-ONE, 1,9-DIHYDRO-9-B-D-RIBOFURANOSYL-"/BI OR
                 691344-25-9/BI OR 740029-83-8/BI OR 9-B-D-RIBOFURANOSYLHYPOX
                 ANTHINE/BI)

=> s L2 and (tissue or neural or spinal)(w)regeneration
L3            2 L2 AND (TISSUE OR NEURAL OR SPINAL)(W) REGENERATION

=> d L3 1-2 ti abs bib

L3   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   A novel, biodegradable polymer conduit delivers neurotrophins and promotes
     nerve regeneration
AB   A wide variety of substances promote neuritic extension after nerve
     injury.  An obstacle to achieving the maximal benefit from these
     substances has been the difficulty in effectively delivering the
     substances over a protracted time course that promotes maximal, directed
     growth.  Delivery of a growth-promoting substance through a biodegradable
     conduit, using materials originally designed for drug delivery
     applications, was hypothesized to promote more robust neural
     regeneration than through conduits lacking the substance.  A
     growth factor-loaded biodegradable nerve guidance conduit was created, and
     in vivo nerve regeneration through the conduit was compared with that
     through conduits lacking the substance.  Inosine, a purine
     analog thought to promote axonal extension following neural injury, was
     loaded into cylindrical polymer foams composed of a lactide-glycolide
     copolymer.  In vitro extravasation of inosine was measured over
     a several-week period using spectrophotometry.  The foams were fashioned
     into single-channel cylindrical nerve guidance conduits via a novel,
     low-pressure injection molding technique.  The conduits were then used to
     bridge 7-mm defects in the rat sciatic nerve.  Control conduits lacking
     inosine were implanted into other animals as controls.  In vitro
     spectrophotometric measurements indicated appreciable leaching of
     inosine from the loaded foams over ≥9 wk.  In vivo, after
     10 wk, a higher percentage cross-sectional area composed of neural tissue
     existed through the inosine-loaded conduits compared with
```

controls. A difference was also found in mean fiber diameter between the 2 groups, with the **inosine**-loaded tubes showing a larger diameter than controls. Thus, a nerve regeneration conduit was successfully created that delivers growth-promoting substances over a protracted time course. In vivo, the presence of **inosine** yielded **neural regeneration** whose histol. features suggest possible superior long-term motor function.

AN 1999:638262 CAPLUS
DN 132:141883
TI A novel, biodegradable polymer conduit delivers neurotrophins and promotes nerve regeneration
AU Hadlock, Tessa; Sundback, Cathryn; Koka, Rahul; Hunter, Daniel; Cheney, Mack; Vacanti, Joseph
CS Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston, MA, 02114, USA
SO Laryngoscope (1999), 109(9), 1412-1416
CODEN: LARYA8; ISSN: 0023-852X
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 2 MEDLINE on STN
TI A novel, biodegradable polymer conduit delivers neurotrophins and promotes nerve regeneration.
AB OBJECTIVE/HYPOTHESIS: A wide variety of substances have been shown to promote neuritic extension after nerve injury. An obstacle to achieving the maximal benefit from these substances has been the difficulty in effectively delivering the substances over a protracted time course that promotes maximal, directed growth. In this study the delivery of a growth-promoting substance through a biodegradable conduit, using materials originally designed for drug delivery applications, was hypothesized to promote more robust **neural regeneration** than through conduits lacking the substance. The objectives of this study were to create a growth factor-loaded biodegradable nerve guidance conduit, and to assess in vivo nerve regeneration through the conduit compared with that through conduits lacking the substance. MATERIALS/METHODS: **Inosine**, a purine analogue thought to promote axonal extension following neural injury, was loaded into cylindrical polymer foams composed of a polylactide-co-glycolide copolymer. First, in vitro extravasation of **inosine** was measured over a several week period using spectrophotometry. Second, the foams were fashioned into single-channel cylindrical nerve guidance conduits via a novel, low-pressure injection molding technique. The conduits were then used to bridge 7-mm defects in the rat sciatic nerve (n = 8). Control conduits lacking **inosine** were implanted into another set of animals as controls (n = 12). RESULTS: In vitro spectrophotometric measurements indicated appreciable leaching of **inosine** from the loaded foams over a period of at least 9 weeks. In the in vivo model, after 10 weeks, a higher percentage cross sectional area composed of neural tissue existed through the **inosine**-loaded conduits compared with controls (mean 44%, SD 7.5% vs. 36%, SD 8.6%, respectively). A difference was also found in mean fiber diameter between the two groups, with the **inosine** -loaded tubes showing a statistically significantly larger diameter than controls ($P < .05$). CONCLUSIONS: A nerve regeneration conduit was successfully created that delivers growth promoting substances over a protracted time course. In an in vivo model, the presence of **inosine**, a purine analogue, yielded **neural regeneration** whose histological features suggest possible superior long-term motor function.
AN 1999428834 MEDLINE
DN PubMed ID: 10499046
TI A novel, biodegradable polymer conduit delivers neurotrophins and promotes nerve regeneration.

AU Hadlock T; Sundback C; Koka R; Hunter D; Cheney M; Vacanti J
CS Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston 02114, USA.. hadlock_t@a1.tch.harvard.edu
SO The Laryngoscope, (1999 Sep) Vol. 109, No. 9, pp. 1412-6.
Journal code: 8607378. ISSN: 0023-852X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
ED Entered STN: 26 Oct 1999
Last Updated on STN: 26 Oct 1999
Entered Medline: 14 Oct 1999

=> sL2 and regeneration
SL2 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s L2 and regeneration
L4 169 L2 AND REGENERATION

=> s L4 and py>2002
L5 25 L4 AND PY>2002

=> s L4 ant L5
MISSING OPERATOR L4 ANT
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s L4 not L5
L6 144 L4 NOT L5

=> dup rem L6
PROCESSING COMPLETED FOR L6
L7 121 DUP REM L6 (23 DUPLICATES REMOVED)

=> s L7 and spinal(w)cord
L8 1 L7 AND SPINAL(W) CORD

=> d L8 ti abs bib

L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
TI **Inosine** stimulates extensive axon collateral growth in the rat corticospinal tract after injury
AB The purine nucleoside **inosine** has been shown to induce axon outgrowth from primary neurons in culture through a direct intracellular mechanism. For this study, we investigated the effects of **inosine** in vivo by examining whether it would stimulate axon growth after a unilateral transection of the corticospinal tract. **Inosine** applied with a minipump to the rat sensorimotor cortex stimulated intact pyramidal cells to undergo extensive sprouting of their axons into the denervated **spinal cord** white matter and adjacent neuropil. Axon growth was visualized by anterograde tracing with biotinylated dextran amine and by immunohistochem. with antibodies to GAP-43. Thus, **inosine**, a naturally occurring metabolite without known side effects, might help to restore essential circuitry after injury to the central nervous system.
AN 1999:765586 CAPLUS
DN 132:73530
TI **Inosine** stimulates extensive axon collateral growth in the rat corticospinal tract after injury
AU Benowitz, Larry I.; Goldberg, David E.; Madsen, Joseph R.; Soni, Deepa;

Irwin, Nina
CS Department of Neurosurgery, Children's Hospital, Harvard Medical School, Boston, MA, 02115, USA
SO Proceedings of the National Academy of Sciences of the United States of America (1999), 96(23), 13486-13490
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s purine and spinal and regeneration
L9 9 PURINE AND SPINAL AND REGENERATION

=> s L9 and py<2003
L10 4 L9 AND PY<2003

=> d L10 1-4 ti abs bib

L10 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
TI Inosine stimulates extensive axon collateral growth in the rat corticospinal tract after injury
AB The **purine** nucleoside inosine has been shown to induce axon outgrowth from primary neurons in culture through a direct intracellular mechanism. For this study, we investigated the effects of inosine in vivo by examining whether it would stimulate axon growth after a unilateral transection of the corticospinal tract. Inosine applied with a minipump to the rat sensorimotor cortex stimulated intact pyramidal cells to undergo extensive sprouting of their axons into the denervated **spinal** cord white matter and adjacent neuropil. Axon growth was visualized by anterograde tracing with biotinylated dextran amine and by immunohistochem. with antibodies to GAP-43. Thus, inosine, a naturally occurring metabolite without known side effects, might help to restore essential circuitry after injury to the central nervous system.
AN 1999:765586 CAPLUS
DN 132:73530
TI Inosine stimulates extensive axon collateral growth in the rat corticospinal tract after injury
AU Benowitz, Larry I.; Goldberg, David E.; Madsen, Joseph R.; Soni, Deepa; Irwin, Nina
CS Department of Neurosurgery, Children's Hospital, Harvard Medical School, Boston, MA, 02115, USA
SO Proceedings of the National Academy of Sciences of the United States of America (**1999**), 96(23), 13486-13490
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
TI Trophic effects of purines in neurons and glial cells
AB A review with many refs. In addition to their well known roles within cells, **purine** nucleotides such as adenosine 5' triphosphate (ATP) and guanosine 5' triphosphate (GTP), nucleosides such as adenosine and guanosine and bases, such as adenine and guanine and their metabolic products xanthine and hypoxanthine are released into the extracellular space where they act as intercellular signaling mols. In the nervous system they mediate both immediate effects, such as neurotransmission, and trophic effects which induce changes in cell metabolism, structure and function and therefore have a longer time course. Some trophic effects of purines are mediated via purinergic cell surface receptors, whereas others

require uptake of purines by the target cells. **Purine** nucleosides and nucleotides, especially guanosine, ATP and GTP stimulate incorporation of [3H]thymidine into DNA of astrocytes and microglia and concomitant mitosis in vitro. High concns. of adenosine also induce apoptosis, through both activation of cell-surface A3 receptors and through a mechanism requiring uptake into the cells. Extracellular purines also stimulate the synthesis and release of protein trophic factors by astrocytes, including bFGF (basic fibroblast growth factor), nerve growth factor (NGF), neurotrophin-3, ciliary neurotrophic factor and S-100β protein. In vivo infusion into brain of adenosine analogs stimulates reactive gliosis. **Purine** nucleosides and nucleotides also stimulate the differentiation and process outgrowth from various neurons including primary cultures of hippocampal neurons and pheochromocytoma cells. A tonic release of ATP from neurons, its hydrolysis by ectonucleotidases and subsequent re-uptake by axons appears crucial for normal axonal growth. Guanosine and GTP, through apparently different mechanisms, are also potent stimulators of axonal growth in vitro. In vivo the extracellular concentration of purines depends on a balance between the release of purines from cells and their re-uptake and extracellular metabolism **Purine** nucleosides and nucleotides are released from neurons by exocytosis and from both neurons and glia by non-exocytotic mechanisms. Nucleosides are principally released through the equilibratory nucleoside transmembrane transporters whereas nucleotides may be transported through the ATP binding cassette family of proteins, including the multidrug resistance protein. The extracellular **purine** nucleotides are rapidly metabolized by ectonucleotidases. Adenosine is deaminated by adenosine deaminase (ADA) and guanosine is converted to guanine and deaminated by guanase. Nucleosides are also removed from the extracellular space into neurons and glia by transporter systems. Large quantities of purines, particularly guanosine and, to a lesser extent adenosine, are released extracellularly following ischemia or trauma. Thus purines are likely to exert trophic effects in vivo following trauma. The extracellular **purine** nucleotide GTP enhances the tonic release of adenine nucleotides, whereas the nucleoside guanosine stimulates tonic release of adenosine and its metabolic products. The trophic effects of guanosine and GTP may depend on this process. Guanosine is likely to be an important trophic effector in vivo because high concns. remain extra-cellularly for up to a week after focal brain injury. **Purine** derivs. are now in clin. trials in humans as memory-enhancing agents in Alzheimer's disease. Two of these, propentofylline and AIT-082, are trophic effectors in animals, increasing production of neurotrophic factors in brain and **spinal** cord. Likely more clin. uses for **purine** derivs. will be found; purines interact at the level of signal-transduction pathways with other transmitters, for example, glutamate. They can beneficially modify the actions of these other transmitters.

AN 1999:678725 CAPLUS
DN 132:102877
TI Trophic effects of purines in neurons and glial cells
AU Rathbone, Michel P.; Middlemiss, Pamela J.; Gysbers, John W.; Andrew, Craig; Herman, Mary A. R.; Reed, Juta K.; Ciccarelli, Renata; Di Iorio, Patrizia; Caciagli, Francesco
CS Department of Medicine, McMaster University, Hamilton, Can.
SO Progress in Neurobiology (Oxford) (**1999**), 59(6), 663-690
CODEN: PGNBA5; ISSN: 0301-0082
PB Elsevier Science Ltd.
DT Journal; General Review
LA English
RE.CNT 347 THERE ARE 347 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
TI Impaired **regeneration** of monoglutamyl tetrahydrofolate leads to cellular folate depletion in mothers affected by a spina bifida pregnancy
AB Periconceptional folate prevents neural tube defects (NTD) by a mechanism

which is unclear. The present study found significant changes in the equilibrium of the homocysteine remethylation cycle in NTD affected mothers, possibly involving B12-dependent methionine synthase or 5,10-methylenetetrahydrofolate reductase. Data were consistent with impaired Hcy remethylation leading to poor **regeneration** of H4PteGlu1, the main intracellular precursor of all folates. This lesion leads to cellular folate deficiency indicated by a significantly lower radioassay RBC folate and 5CH3H4PteGlu4 in affected mothers. The drop in this tetraglutamate is associated with an increase in the abundance of longer chain oligo-γ-glutamyl folate, again reflecting the underlying folate deficiency. This effect may compromise **purine**, DNA-thymine, and methionine production, particularly during embryogenesis when folate demand is high. At this time serine hydroxymethyltransferase may play a critical role in conserving H4PteGlu1 for **purine** synthesis. Many of these depletion effects were corrected with folate supplementation for 1 mo. (c) 1998 Academic Press.

AN 1998:726912 CAPLUS
DN 130:80783
TI Impaired **regeneration** of monoglutamyl tetrahydrofolate leads to cellular folate depletion in mothers affected by a spina bifida pregnancy
AU Lucock, M. D.; Daskalakis, I.; Lumb, C. H.; Schorah, C. J.; Levene, M. I.
CS Research School of Medicine, Centre for Reproduction, Growth and Development, Division of Paediatrics and Child Health, Leeds General Infirmary, University of Leeds, Leeds, LS2 9NS, UK
SO Molecular Genetics and Metabolism (**1998**), 65(1), 18-30
CODEN: MGMEFF; ISSN: 1096-7192
PB Academic Press
DT Journal
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 4 MEDLINE on STN
TI Impaired **regeneration** of monoglutamyl tetrahydrofolate leads to cellular folate depletion in mothers affected by a spina bifida pregnancy.
AB Periconceptional folate prevents neural tube defects (NTD) by a mechanism which is unclear. The present study found significant changes in the equilibrium of the homocysteine remethylation cycle in NTD affected mothers, possibly involving B12-dependent methionine synthase or 5,10-methylenetetrahydrofolate reductase. Data were consistent with impaired Hcy remethylation leading to poor **regeneration** of H4PteGlu1, the main intracellular precursor of all folates. This lesion leads to cellular folate deficiency indicated by a significantly lower radioassay RBC folate and 5CH3H4PteGlu4 in affected mothers. The drop in this tetraglutamate is associated with an increase in the abundance of longer chain oligo-gamma-glutamyl folate, again reflecting the underlying folate deficiency. This effect may compromise **purine**, DNA-thymine, and methionine production, particularly during embryogenesis when folate demand is high. At this time serine hydroxymethyltransferase may play a critical role in conserving H4PteGlu1 for **purine** synthesis. Many of these depletion effects were corrected with folate supplementation for 1 month.

AN 1999005505 MEDLINE
DN PubMed ID: 9787091
TI Impaired **regeneration** of monoglutamyl tetrahydrofolate leads to cellular folate depletion in mothers affected by a spina bifida pregnancy.
AU Lucock M D; Daskalakis I; Lumb C H; Schorah C J; Levene M I
CS Research School of Medicine, Centre for Reproduction, Growth and Development, Division of Paediatrics and Child Health, D Floor, Clarendon Wing, Leeds General Infirmary, University of Leeds, Leeds, West Yorkshire, LS2 9NS, United Kingdom.
SO Molecular genetics and metabolism, **(1998 Sep)** Vol. 65, No. 1, pp. 18-30.
Journal code: 9805456. ISSN: 1096-7192.

```
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   199811
ED   Entered STN: 6 Jan 1999
     Last Updated on STN: 6 Jan 1999
     Entered Medline: 25 Nov 1998


=> file registry
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                     162.55     168.29

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                      -3.75      -3.75

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STRUCTURE FILE UPDATES:   30 APR 2006  HIGHEST RN 882297-19-0
DICTIONARY FILE UPDATES:  30 APR 2006  HIGHEST RN 882297-19-0

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* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,   *
* effective March 20, 2005.  A new display format, IDERL, is now    *
* available and contains the CA role and document type information. *
*                                                                   *
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predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> s guanosine/cn
L11           1 GUANOSINE/CN

=> sel L11
E28 THROUGH E40 ASSIGNED

=> s adenosine/cn
L12           1 ADENOSINE/CN
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=> sel L12
E41 THROUGH E64 ASSIGNED

=> file caplus medline
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                             ENTRY    SESSION
FULL ESTIMATED COST                                          10.62     178.91

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)              SINCE FILE      TOTAL
                                                             ENTRY    SESSION
CA SUBSCRIBER PRICE                                           0.00      -3.75

FILE 'CAPLUS' ENTERED AT 16:41:37 ON 01 MAY 2006
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FILE 'MEDLINE' ENTERED AT 16:41:37 ON 01 MAY 2006

=> s E28-E64
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
COMMAND INTERRUPTED
If this message appears repeatedly, please notify the Help Desk.
Enter "HELP STN" for information on contacting the nearest STN Help
Desk by telephone or via SEND in the STNMAIL file.

=> s E28-E40
L13       54097 ("B-D-RIBOFURANOSIDE, GUANINE-9"/BI OR "GUANINE RIBONUCLEOS
                 IDE"/BI OR "GUANINE, 9-B-D-RIBOFURANOSYL-"/BI OR GUANOSINE/
                BI OR "INOSINE, 2-AMINO-"/BI OR "NSC 19994"/BI OR VERNINE/BI OR
                118-00-3/BI OR "2-AMINO-1,9-DIHYDRO-9-B-D-RIBOFURANOSYL-6H-P
                URIN-6-ONE"/BI OR 484-80-0/BI OR "6H-PURIN-6-ONE, 2-AMINO-1,9-DIH
                YDRO-9-B-D-RIBOFURANOSYL-"/BI OR 685891-87-6/BI OR 85-30-3/B
                I)

=> s E41-E64
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
   1 FILES SEARCHED...
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
COMMAND INTERRUPTED
If this message appears repeatedly, please notify the Help Desk.
Enter "HELP STN" for information on contacting the nearest STN Help
Desk by telephone or via SEND in the STNMAIL file.

=> s (E41-E64 and regeneration and spinal)
L14        4738 ((B-ADENOSINE/BI OR B-D-ADENOSINE/BI OR "B-D-RIBO
                 FURANOSE, 1-(6-AMINO-9H-PURIN-9-YL)-1-DEOXY-"/BI OR "B-D-RI
                BOFURANOSIDE, ADENINE-9"/BI OR A/BI OR "ADENINE RIBOSIDE"/BI OR
                ADENOCARD/BI OR ADENOCOR/BI OR ADENOSCAN/BI OR ADENOSINE/BI OR
                ADREKAR/BI OR BONITON/BI OR D-ADENOSINE/BI OR MYOCOL/BI OR "NSC
                7652"/BI OR NUCLEOCARDYL/BI OR RIBOADENOSINE/BI OR SANDESIN/BI
                OR 46946-45-6/BI OR 46969-16-8/BI OR 58-61-7/BI OR 9-B-D-RIB
                OFURANOSYL-9H-PURIN-6-AMINE/BI OR 9-B-D-RIBOFURANOSYLADENINE
                /BI OR "9H-PURIN-6-AMINE, 9-B-D-RIBOFURANOSYL-"/BI) AND
                REGENERATION AND SPINAL)

=> s (L14 and regeneration and spinal)
L15        4738 (L14 AND REGENERATION AND SPINAL)

=> s (L13 and regeneration and spinal)
L16           6 (L13 AND REGENERATION AND SPINAL)

=> s L15 or L16
L17        4738 L15 OR L16
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=> s L17 and py>2002
L18       1458 L17 AND PY>2002

=> s L17 not L18
L19       3280 L17 NOT L18

=> dup rem L19
PROCESSING IS APPROXIMATELY  87% COMPLETE FOR L19
PROCESSING COMPLETED FOR L19
L20        2687 DUP REM L19 (593 DUPLICATES REMOVED)

=> s L20 and (spinal(w)cord)
L21       1842 L20 AND (SPINAL(W) CORD)

=> s L21 and injury
L22        784 L21 AND INJURY

=> s L22 and electrical(w)stimulation
L23         13 L22 AND ELECTRICAL(W) STIMULATION

=> d L23 1-13 ti

L23  ANSWER 1 OF 13      MEDLINE on STN
TI   Recent advances in pathophysiology and treatment of spinal
     cord injury.

L23  ANSWER 2 OF 13      MEDLINE on STN
TI   Spontaneous reconstruction of the canine hypogastric nerve over a
     long period after removing half of its length.

L23  ANSWER 3 OF 13      MEDLINE on STN
TI   The intercostal to phrenic nerve transfer: an effective means of
     reanimating the diaphragm in patients with high cervical spine
     injury.

L23  ANSWER 4 OF 13      MEDLINE on STN
TI   Alterations in the distribution of stimulus-evoked c-fos in the
     spinal cord after neonatal peripheral nerve
     injury in the rat.

L23  ANSWER 5 OF 13      MEDLINE on STN
TI   Using a direct current electrical field to promote
     spinal-cord regeneration.

L23  ANSWER 6 OF 13      MEDLINE on STN
TI   "Skin-CNS-bladder" reflex pathway for micturition after spinal
     cord injury and its underlying mechanisms.

L23  ANSWER 7 OF 13      MEDLINE on STN
TI   Advances in spinal cord regeneration.

L23  ANSWER 8 OF 13      MEDLINE on STN
TI   Repair, reconstruction, regeneration and rehabilitation
     strategies to spinal cord injury.

L23  ANSWER 9 OF 13      MEDLINE on STN
TI   Regenerated dorsal root fibers form functional synapses in embryonic
     spinal cord transplants.

L23  ANSWER 10 OF 13      MEDLINE on STN
TI   Characterization of longitudinal field gradients from electrical
     stimulation in the normal and injured rodent spinal
     cord.
```

L23 ANSWER 11 OF 13 MEDLINE on STN
TI The growth-associated protein GAP-43 appears in dorsal root ganglion cells and in the dorsal horn of the rat **spinal cord** following peripheral nerve **injury.**

L23 ANSWER 12 OF 13 MEDLINE on STN
TI Pathology of **spinal** injuries.

L23 ANSWER 13 OF 13 MEDLINE on STN
TI Effect of peripheral nerve **injury** on receptive fields of cells in the cat **spinal cord.**

=> d L23 1 5 6 7 8 9 10 12 ti abs bib

L23 ANSWER 1 OF 13 MEDLINE on STN
TI Recent advances in pathophysiology and treatment of **spinal cord injury.**
AB Thirty years ago, patients with **spinal cord injury** (SCI) and their families were told "nothing can be done" to improve function. Since the SCI patient population is reaching normal life expectancy through better health care, it has become an obviously worthwhile enterprise to devote considerable research effort to SCI. Targets for intervention in SCI toward improved function have been identified using basic research approaches and can be simplified into **a** list: (1) reduction of edema and free-radical production, (2) rescue of neural tissue at risk of dying in secondary processes such as abnormally high extracellular glutamate concentrations, (3) control of inflammation, (4) rescue of neuronal/glial populations at risk of continued apoptosis, (5) repair of demyelination and conduction deficits, (6) promotion of neurite growth through improved extracellular environment, (7) cell replacement therapies, (8) efforts to bridge the gap with transplantation approaches, (9) efforts to retrain and relearn motor tasks, (10) restoration of lost function by **electrical stimulation**, and (11) relief of chronic pain syndromes. Currently, over 70 clinical trials are in progress worldwide. Consequently, in this millennium, unlike in the last, no SCI patient will have to hear "nothing can be done."
AN 2002682412 MEDLINE
DN PubMed ID: 12443996
TI Recent advances in pathophysiology and treatment of **spinal cord injury.**
AU Hulsebosch Claire E
CS Department of Anatomy and Neurosciences, Marine Biomedical Institute, University of Texas Medical Branch, Galveston, Texas 77555-1043, USA.. cehulseb@utmb.edu
NC NS-11225 (NINDS)
NS-39161 (NINDS)
SO Advances in physiology education, (2002 Dec) Vol. 26, No. 1-4, pp. 238-55. Ref: 154
Journal code: 100913944. ISSN: 1043-4046.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200306
ED Entered STN: 22 Nov 2002
Last Updated on STN: 12 Jun 2003
Entered Medline: 11 Jun 2003

L23 ANSWER 5 OF 13 MEDLINE on STN
TI Using **a** direct current electrical field to promote **spinal-cord regeneration.**
AB The authors used **a** direct current electrical field to promote

spinal-cord regeneration in **a** canine model. Thirty-two dogs were randomly divided into four groups. Complete **spinal-cord injury** was induced, and electrical stimulators were then placed in the animals. Group 1 served as controls; Groups 2 to 4 were experimental groups, with varying stimulator voltages: 0V in Group 1, 12V in Groups 2 and 4, and 6V in Group 3, with the stimulator implanted 6 hr after **spinal-cord injury** in Group 4. Functional, electrophysiologic and morphometric assessments were carried out 1 to 3 months postoperatively. Results showed that **spinal-cord** function, cortical somatosensory evoked potentials, number of neurons, sectional area of neurons, and Nissl body density in the experimental groups were much better than those in the control group. In addition, all the indices in Group 2 were better than those in Groups 3 and 4. This indicated that direct current **electrical stimulation** could effectively promote **spinal-cord regeneration** and functional recovery in this model. The 12V voltage was safe for the animals. The stimulator was not rejected by the host for **a** relatively long period of time.

AN 1999408439 MEDLINE
DN PubMed ID: 10480562
TI Using **a** direct current electrical field to promote **spinal-cord regeneration.**
AU Shen N J; Wang S C
CS Department of Orthopedics, People's Hospital of Hainan Province, Haikou, China.
SO Journal of reconstructive microsurgery, (1999 Aug) Vol. 15, No. 6, pp. 427-31.
Journal code: 8502670. ISSN: 0743-684X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
ED Entered STN: 26 Oct 1999
Last Updated on STN: 26 Oct 1999
Entered Medline: 14 Oct 1999

L23 ANSWER 6 OF 13 MEDLINE on STN
TI "Skin-CNS-bladder" reflex pathway for micturition after **spinal cord injury** and its underlying mechanisms.
AB PURPOSE: **A** "skin-CNS-bladder" reflex pathway for inducing micturition after **spinal cord injury** has been established in cat. This reflex pathway which is basically **a** somatic reflex arc with **a** modified efferent limb that passes somatic motor impulses to the bladder, has been designed to allow **spinal cord** injured patients to initiate voiding by scratching the skin. MATERIALS AND METHODS: The skin-CNS-bladder reflex was established in the cat by intradural microanastomosis of the left L7 ventral root (VR) to the S1 VR while leaving the L7 dorsal root (DR) intact to conduct cutaneous afferent signals that can trigger the new micturition reflex arc. After allowing 11 weeks for axonal **regeneration**, urodynamic, pharmacological and electrophysiological studies were conducted in pentobarbital or chloralose anesthetized animals. RESULTS: **A** detrusor contraction was initiated at short latency by scratching the skin or by percutaneous **electrical stimulation** in the L7 dermatome. Maximal bladder pressures during this stimulation were similar to those activated by bladder distension in control animals. Electrophysiological recording revealed that single stimuli (0.3 to 3 mA, 0.02 to 0.2 msec duration) to the left L7 **spinal** nerve in which the efferent axons had degenerated evoked action potentials (0.5 to 1 mV) in the left S1 **spinal** nerve distal to the anastomosis. In addition, increases in bladder pressure were elicited by trains of the stimuli (5 to 20 Hz, 5 seconds) applied to the L7 **spinal** nerve. Urodynamic studies including external

sphincter EMG recording demonstrated that the new reflex pathway could initiate voiding without detrusor-external urethral sphincter dyssynergia. Atropine (0.05 mg./kg., i.v.) or trimethaphan (5 mg./kg., i.v.), **a** ganglionic blocking agent, depressed the bladder contractions elicited by skin stimulation. The skin-CNS-bladder reflex could also be elicited after transecting the **spinal cord** at the L2-L3 or L7-S1 levels. CONCLUSION: The cross-wired somato-autonomic bladder reflex is effective in initiating bladder contractions and coordinated voiding in cats with an intact neuraxis and can also induce bladder contractions after acute transection of the lumbar **spinal cord.** The new pathway is mediated by cholinergic transmission involving both nicotinic and muscarinic receptors. It is concluded that somatic motor axons can innervate bladder parasympathetic ganglion cells and thereby transfer somatic reflex activity to the bladder smooth muscle.

AN 1999385594 MEDLINE
DN PubMed ID: 10458412
TI "Skin-CNS-bladder" reflex pathway for micturition after **spinal cord injury** and its underlying mechanisms.
AU Xiao C G; de Groat W C; Godec C J; Dai C; Xiao Q
CS Department of Urology, the Long Island College Hospital, SUNY Health Science Center at Brooklyn, New York 11203, USA.
NC R01 DK 44877-01 (NIDDK)
SO The Journal of urology, (1999 Sep) Vol. 162, No. 3 Pt 1, pp. 936-42. Journal code: 0376374. ISSN: 0022-5347.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199909
ED Entered STN: 5 Oct 1999
Last Updated on STN: 5 Oct 1999
Entered Medline: 23 Sep 1999

L23 ANSWER 7 OF 13 MEDLINE on STN
TI Advances in **spinal cord regeneration.**
AB **Spinal cord injury** continues to be **a** major cause of morbidity, particularly among young people involved in vehicle-related trauma, falls, and sports injuries. Although research advances are still **a** long way from clinical treatments, recent studies on animals have indicated new possibilities for recovery of function. In this review, these new findings on the use of neurotrophic factors, antibodies to inhibitory molecules, **electrical stimulation**, and transplantation of peripheral nerves and olfactory glial cells, and their success in achieving functional recovery after adult **spinal cord** lesions are discussed.
AN 1999259504 MEDLINE
DN PubMed ID: 10327520
TI Advances in **spinal cord regeneration.**
AU Lu J; Waite P
CS Neural Injury Research Unit, School of Anatomy, University of New South Wales.
SO Spine, (1999 May 1) Vol. 24, No. 9, pp. 926-30. Ref: 68
Journal code: 7610646. ISSN: 0362-2436.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 14 Jul 1999
Last Updated on STN: 14 Jul 1999
Entered Medline: 28 Jun 1999

L23 ANSWER 8 OF 13 MEDLINE on STN
TI Repair, reconstruction, **regeneration** and rehabilitation

strategies to **spinal cord injury.**

AB The structural changes seen in the transected **spinal cord** followed by transplantation of the distal ends (neuroma) of intercostal nerve inserted into the **spinal cord** proximal and distal to the transection lesion site. This activates CNS axonal **regeneration.** 2,3,4 These changes refer to the plasticity in the nervous system following damage to the **spinal cord.** There is **regeneration** and growth and synapotogenesis and remodeling of synaptic connections, development of reflex activity in the denervated cord. Nerve growth factors and neurotrophic factors sustain and maintain **a** degree of functional integrity of structural neural circuitry. 2,3,4,13 The end result is standing, stepping, and reflex walking in 28 female mature dogs. 2,3,4,5 **Electrical stimulation** of the anastomosed intercostal nerves resulted in hind limb movements and recording of the electromyograms of the contracting muscles. Twenty-six control dogs and animals with behavioral depression are unable to follow rehabilitative procedures developed muscle atrophy, ankylosis of joints, decrease in bone density, decrease in reflex activity of the **spinal cord** distal to the transection. 2,3,4,5

AN 1998265775 MEDLINE

DN PubMed ID: 9603065

TI Repair, reconstruction, **regeneration** and rehabilitation strategies to **spinal cord injury.**

AU Turbes C C

CS Department of Biomedical Sciences, Creighton University, Omaha, NE 68178, USA.

SO Biomedical sciences instrumentation, (1997) Vol. 34, pp. 351-6. Journal code: 0140524. ISSN: 0067-8856.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199806

ED Entered STN: 13 Jul 1998
Last Updated on STN: 13 Jul 1998
Entered Medline: 26 Jun 1998

L23 ANSWER 9 OF 13 MEDLINE on STN

TI Regenerated dorsal root fibers form functional synapses in embryonic **spinal cord** transplants.

AB 1. The aim of the present study was to determine whether synapses formed by dorsal root afferents that regenerate into intraspinal transplants of fetal **spinal cord** are functional. Severed L4 or L5 dorsal root stumps were placed at the bottom of dorsal quadrant cavities made in the lumbar **spinal** cords of adult rats and juxtaposed to embryonic day 14 **spinal cord** transplants. 2. In animals examined 5-10 weeks later, we recorded extracellularly in transplants from 43 units that fired in response to **electrical stimulation** of the implanted dorsal root. Latency fluctuations of extracellular firing that increase with stimulus and failure to follow high-frequency and posttetanic potentiation of extracellular firing stimulation suggest that synapses with conventional properties are formed between regenerating afferents and transplant neurons. Limited intracellular recordings confirmed the existence of excitatory postsynaptic potentials in transplant neurons after dorsal root stimulation. 3. In 16 units, extracellular firing occurred in response to single shock stimulation. The remainder of the units required two or more dorsal root shocks to evoke firing; some of these connections also may be monosynaptic. 4. Under the assumption that single shock firing was most likely the result of monosynaptic connections between transplant neurons and regenerated dorsal root fibers, we estimated the conduction velocities of regenerated fibers. These estimates suggest that fibers with conduction velocities in the C, **A** delta, and **A** alpha/beta ranges regenerate into transplants of embryonic **spinal**

cord. 5. The results demonstrate that regenerated dorsal root axons establish functional synaptic connections with transplant neurons. The implications for using fetal transplants to help rebuild **spinal** reflex circuits after **spinal cord injury** are considered.

AN 97025009 MEDLINE
DN PubMed ID: 8871233
TI Regenerated dorsal root fibers form functional synapses in embryonic **spinal cord** transplants.
AU Itoh Y; Waldeck R F; Tessler A; Pinter M J
CS Department of Anatomy and Neurobiology, Medical College of Pennsylvania, Philadelphia 19129, USA.
NC NS-24707 (NINDS)
SO Journal of neurophysiology, (1996 Aug) Vol. 76, No. 2, pp. 1236-45. Journal code: 0375404. ISSN: 0022-3077.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 26 Mar 1998
Last Updated on STN: 26 Mar 1998
Entered Medline: 16 Mar 1998

L23 ANSWER 10 OF 13 MEDLINE on STN
TI Characterization of longitudinal field gradients from **electrical stimulation** in the normal and injured rodent **spinal cord.**
AB The purpose of this experiment was to characterize the longitudinal field gradients from exogenously applied **electrical stimulation** in the normal and injured rodent **spinal cord.** In addition, we compared the field gradients arising from stimulation with two different types of stimulating electrodes. Twenty normal rats underwent the surgical implantation of either extradural disc (n = 10) or cuff (n = 10) electrodes in the lower cervical and upper thoracic **spinal cord.** Sine waves of 1.5 to 50 microA and 0.5 to 50 Hz were used for stimulation. Field gradients were measured differentially from two extracellular glass microelectrodes, positioned stereotactically in the **spinal cord** at different locations between the stimulating electrodes. The effect of acute **spinal cord injury** on local field strength was studied in five animals from each group. The field gradients from stimulation with disc electrodes were greatest in close proximity to the discs and decreased markedly toward the point equidistant between the electrodes. In contrast, the gradients produced by cuff electrodes were much more evenly distributed along the **spinal cord,** increasing only slightly in proximity to the electrodes. These fields were also more evenly distributed throughout the **spinal cord** in cross-section but were generally weaker than those arising from disc electrodes. Acute **spinal cord injury** significantly increased the field gradients arising from both disc and cuff electrodes. However, the observed gradients remained substantially lower than those reported to enhance neurite growth in vitro. We conclude that the position and design of stimulating electrodes has **a** profound effect on longitudinal field gradients within the mammalian **spinal cord,** as does the presence of an acute **spinal cord injury.**
AN 94247580 MEDLINE
DN PubMed ID: 8190223
TI Characterization of longitudinal field gradients from **electrical stimulation** in the normal and injured rodent **spinal cord.**
AU Hurlbert R J; Tator C H
CS Playfair Neuroscience Unit, Toronto Hospital, Ontario, Canada.
SO Neurosurgery, (1994 Mar) Vol. 34, No. 3, pp. 471-82; discussion 482-3.

Journal code: 7802914. ISSN: 0148-396X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199406
ED Entered STN: 29 Jun 1994
Last Updated on STN: 29 Jun 1994
Entered Medline: 17 Jun 1994

L23 ANSWER 12 OF 13 MEDLINE on STN
TI Pathology of **spinal** injuries.
AB Information about the neuropathology of **spinal cord injury** is derived from the personal study of 341 specimens; 225 of these were traumatic, including 123 with massive injuries. Thirty-one were associated with metastases, 38 were degenerative, and 6 were infectious. Included are 41 normal controls. The hyperacute human **spinal injury** study provides **a** reference base for animal experiments. **A** sound knowledge of the pathology of **spinal injury** is an essential prerequisite for the competent management of patients with these injuries. Because no lesions were found that would be amenable to surgical correction, the study supports the conservative approach, in keeping with the teaching and practice of Sir Ludwig Guttmann and Sir George Bedbrook. In most specimens traumatic necrosis was most severe in the central gray matter and adjoining posterior columns of the cord. Preserved continuity of **a** proportion of the lateral, anterior, and posterior white matter was usual. Space-taking subdural or extradural hemorrhages and hematomyelia were rare. In patients who survived for more than **a** few weeks, posttraumatic cysts resulted from removal of necrotic parenchyma by macrophages. Although in very severe injuries complete disruption of both bony and **spinal cord** tissues was observed, others with equally massive injuries showed some continuity of the **spinal cord** parenchyma. This somewhat unexpected observation is in accord with physiologic studies in which poly EMG and sensory-evoked potentials demonstrate continuity of long tracts across the lesion in patients who were otherwise clinically complete. **Regeneration** of nerve roots and to **a** lesser extent of central axons was evident in patients who survived for more than 5 or 6 months. Complications consisted of ascending or descending necrosis and enlarging cavities. There is clinical and physiologic evidence of remodeling of reflex systems in the **spinal** patient that manifests as **a** changing neurologic picture. It is possible that the use of **a** variety of techniques, such as **electrical stimulation**, would influence such plastic changes to the benefit of the patient. Little detailed anatomic information is available on this topic as **a** key area for future investigation.
AN 86133611 MEDLINE
DN PubMed ID: 6545680
TI Pathology of **spinal** injuries.
AU Kakulas B A
SO Central nervous system trauma : journal of the American Paralysis Association, (1984 Winter) Vol. 1, No. 2, pp. 117-29.
Journal code: 8501356. ISSN: 0737-5999.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198604
ED Entered STN: 21 Mar 1990
Last Updated on STN: 21 Mar 1990
Entered Medline: 23 Apr 1986

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